Biological action of pancreatic amylin: relationship with glucose metabolism, diabetes, obesity and calcium metabolism.

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Amylin, also called islet amyloid polypeptide (IAPP), or diabetes-associated peptide (DAP) is a recently discovered 37 amino acid polypeptide which has been shown to be co-secreted with insulin from the pancreatic beta-cell. The peptide turned out to be the major constituent of pancreatic amyloid deposits which are frequently found in the pancreas of type II diabetic patients. Therefore, a role for amylin in the aetiology of type II diabetes was hypothesized. To investigate this possibility, several studies have been performed to elucidate whether amylin is able to impair insulin secretion and action, two characteristic features of type II diabetes mellitus. These studies suggest that it is unlikely that amylin has a direct inhibitory effect on insulin secretion. Amyloid deposits, however, which are derived from the in situ polymerization and precipitation of amylin, may impair beta-cell function during type II diabetes by damaging and covering beta-cells. Furthermore, it has been shown that amylin has the potential to antagonize the action of insulin on glucose metabolism by increasing hepatic glucose production and by decreasing muscle, but not adipocyte glucose uptake. For these reasons, it has been suggested that amylin might be involved in the pathophysiology of type II diabetes and obesity, disease states which are characterized by abnormal beta-cell function and insulin resistance. In addition, amylin

was shown to induce hypocalcaemia by inhibiting osteoclast-mediated bone resorption in a calcitonin-like manner. Therefore, amylin is likely to be involved in both the modulation of glucose and calcium metabolism.

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